AMERICAN JOURNAL OF PHYSICAL ANTHROPOLOGY 102:133–139 (1997)

# Dental Asymmetry, Maternal Obesity, and Smoking

JULES A. KIESER,[1]* HENNIE T. GROENEVELD,[3] AND PAULA C.F. DA SILVA[2]
[1]*Department of Oral Biology, School of Dentistry, Otago University, Dunedin, New Zealand;* [2]*Department of Oral Pathology, School of Dentistry, Witwatersrand University, Johannesburg, South Africa;* [3]*Department of Statistics, University of Pretoria, Pretoria, South Africa*

*KEY WORDS* fluctuating asymmetry, obesity, maternal smoking

*ABSTRACT* This study examines the levels of fluctuating dental asymmetry in four samples of school children: those whose mothers were obese and had smoked during the pregnancy concerned (n = 111); those whose mothers were obese non-smokers (n = 114); those whose mothers were non-obese smokers (n = 104); and those whose mothers were lean non-smokers (n = 111). The degree of fluctuating asymmetry was assessed by means of a rescaled asymmetry measure. Obesity was defined as Quetelet's index in excess of 30, and smoking status as at least 20 cigarettes per day during the pregnancy concerned. When the magnitudes of fluctuating asymmetry in children of lean smokers were compared to the control group of lean nonsmokers, no significant univariate differences were found. Children of obese mothers, whether these smoked or not, were found to have significantly raised levels of asymmetry. An analysis of variance confirmed that the combination of obesity and maternal smoking was a significant predictor of fluctuating dental asymmetry. The teeth involved tended to be the maxillary first incisor and molars. It is concluded that maternal obesity has a destabilizing effect on the developing fetus and that this effect appears to be enhanced in obese mothers who smoked. This effect was absent in lean mothers, irrespective of their smoking status. Am J Phys Anthropol 102:133–139. 

Developmental destabilization may be inferred from a determination of the levels of fluctuating asymmetry of a given bilateral trait (Van Valen, 1962; Palmer and Strobeck, 1986; Parson, 1990; Livshits and Kobyliansky, 1991; Zakharov, 1992). Such studies have provided valuable information on host responses to thermal, audiogenic, dietary and thermal stresses in laboratory animals (e.g. Siegel and Doyle, 1975; Gest et al., 1982; Mooney et al., 1985; Kohn and Bennett, 1986). Levels of dental fluctuating asymmetry have been used to quantify various environmental stressors in human populations (Harris and Nweeia, 1980; Townsend, 1981; Kieser et al., 1986; Kieser and Groeneveld, 1988; Livshits and Kobyliansky, 1989; Fraser, 1994). The magnitude of fluctuating asymmetry in humans has also been related to consanguinity (Suarez, 1974; Di Bennardo and Bailit, 1978), to chromosomal aneuploidy (Townsend, 1983; Townsend et al., 1986) and clefting of the lip and palate (Sofaer, 1979). From their recent study of the Bedouin, Hershkovitz and coworkers have shown that fluctuating dental asymmetry was a function of environmental stress (Hershkovitz et al., 1993). In short, fluctuating asymmetry has been shown to be a useful early warning system of developmen-

*Correspondence to: Jules A. Kieser, Department of Oral Pathology, School of Dentistry, Otago University, Dunedin, New Zealand.

Received 5 December 1994; Accepted 3 September 1996.

tal destabilization during ontogeny in a range of species and conditions.

The study of the adverse effects of maternal obesity on the outcome of pregnancy has been pursued for the past 50 years (Mathews and Der Funcke, 1938; Douglas and Scadron, 1951; Petry, 1956; Witten, 1958). This body of literature has firmly established that overweight women show significantly raised incidences of gestational hypertension, inadequate pregnancy weight gain, miscarriages, post partum infections and neonatal macrosomia (Harrison et al., 1980; Kliegman and Gross, 1985; Lucas et al., 1988; Tilton et al., 1988; Naeye, 1990; Hamilton-Fairley et al., 1992). The usual explanation offered has been that obese women tend to be hypertensive, androgenic and diabetic, and also that mechanical factors during the birth process may have an adverse effect on delivery in obese women (Gross et al., 1980; Harrison et al., 1980; Longcope et al., 1986). Recently, however, the possibility has been raised that this observed increase in perinatal morbidity might be systematically related to maternal body weight (Naeye, 1990; Hamilton-Fairley et al., 1992). In other words, there appears to be at least some degree of fetal destabilization in obese women.

In the present paper we examine the levels of dental fluctuating asymmetry in children of obese and non-obese mothers, the aim being to determine whether maternal obesity results in significantly raised levels of fluctuating asymmetry. We also examine the possible effects of maternal cigarette smoking in obese and non-obese mothers.

## MATERIALS AND METHODS

The data for the present investigation were derived from the artificial stone casts of the maxillary arcades of 440 caucasian school children between the ages of 10 and 16 years. Four groups were investigated: 1) those whose mothers were obese and had smoked during the pregnancy concerned (n = 111); 2) those whose mothers were obese non-smokers (n = 114); 3) those whose mothers were non-obese smokers (n = 104); and 4) those whose mothers were non-obese non-smokers (n = 111). The latter (group 4) are the controls.

Obesity was defined as Quetelet's index (weight [kg]/height$^2$ [m]) in excess of 30 (Lucas et al., 1988) and was determined by interview. Smoking was defined as at least 20 cigarettes smoked per day during the pregnancy concerned. The children chosen were all upper-middle-class individuals who had no known congenital abnormalities. The entire procedure, including possible discomforts and benefits, was fully explained to each subject, and informed consent was obtained before each interview.

To determine the accuracy of weight recall, the stated body weights of 21 mothers were compared to actual weights obtained from hospital archives. Paired *t*-tests showed that body weight was under-reported on average 2.09 kg ($P < 0.001$). A Pearson's correlation coefficient of 0.98 ($P < 0.001$) confirmed the close conformance of reported to actual weights.

Mesiodistal (MD) and buccolingual (BL) diameters were determined by one observer (JAK) on the left and right sides of the maxillary dental arcade of each individual, using a sliding caliper on which the tips had been sharpened. No measurements were attempted on carious, worn, or fractured teeth. All models were cast from standard orthodontic impressions in artificial stone and measurements were conducted by one observer (JAK), according to the method described by Kieser et al. (1990). Measurement error was evaluated by means of 40 repeated measurements and yielded a Pearson correlation coefficient of 0.996. Bias or directionality of the error was evaluated by means of a Student's *t*-test and gave a non-significant value of 0.31. A standard rescaled antimeric asymmetry measure (d*) was used because it eliminates the effect of individual size differences among teeth. Here, the absolute side difference is divided by the mean size of the left and right teeth $d^* = [R - L]/[R + L)/2]$ (Harris and Nweeia, 1980).

Within each tooth district, only the first permanent tooth was measured, the reason being that these teeth are better canalized than later developing teeth and, hence, would provide a more rigorous measure of

*TABLE 1. Fluctuating dental asymmetry attributable to measurement error in each of the four classes of children observed*

| Tooth | Dimension | Maternal smoking group: Obese smokers AAME (*P*) | Obese non-smokers AAME (*P*) | Non-obese smokers AAME (*P*) | Non-obese non-smokers[1] AAME (*P*) |
|---|---|---|---|---|---|
| I1 | MD | 0.001 (0.911) | 0.001 (0.866) | 0.001 (0.998) | 0.001 |
| | BL | 0.001 (0.577) | 0.002 (0.439) | 0.001 (0.180) | 0.003 |
| C | MD | 0.001 (0.877) | 0.001 (0.730) | 0.002 (0.878) | 0.002 |
| | BL | 0.003 (0.731) | 0.002 (0.909) | 0.001 (0.644) | 0.002 |
| M1 | MD | 0.001 (0.539) | 0.002 (0.463) | 0.003 (0.981) | 0.001 |
| | BL | 0.002 (0.614) | 0.001 (0.606) | 0.001 (0.594) | 0.001 |

AAME = asymmetry attributable to measurement error; I1 = central maxillary incisor; C = maxillary canine; M1 = first maxillary molar; MD = mesiodistal; BL = buccolingual. Significance of difference from status 4 (non-obese, non-smoker) as measured by Student's *t*-tests is given in parentheses. [1]Control group.

differences in fluctuating asymmetry (Townsend, 1981; Butler, 1983; Kieser, 1990).

To evaluate the impact of measurement error on the observed levels of fluctuating asymmetry, teeth on the left side of each cast were remeasured, and were considered to be a set of "antimeric" teeth (Kieser and Groeneveld, 1994). Differences between these two sets of measurements were evaluated using the asymmetry formula and "asymmetries" due to measurement error were calculated for each of the four categories of children.

## RESULTS

Fluctuating asymmetry attributable to measurement error in each of the classes of children is summarized in Table 1. There were no significant differences between the obese and/or smoking groups and the control group in which mothers were non-obese, non-smokers. We concluded therefore, that the contribution of measurement error to our estimates of asymmetry had not biased our results in any of the four groups investigated.

Mean rescaled asymmetry values for MD and BL dimensions of the maxillary teeth in the sons and daughters of women in all four categories are listed in Table 2. Student's *t*-tests suggest a decided lack of gender dimorphism. The general patterning of asymmetry suggests that MD dimensions are less asymmetrical than BL dimensions.

Table 3 lists asymmetry values for each of the four groups of children (genders pooled). The children of lean smoking mothers were not significantly more asymmetrical than those of controls, lean non-smoking mothers. Significant elevation in fluctuating asymmetry was noted in both the children of obese non-smokers and those of obese smokers. Across the dental arcade, the canine appeared to be the most canalized tooth (Figs. 1 and 2). In other words, levels of asymmetry tended to be higher in the first incisor and molar teeth. The buccolingual diameter showed greater elevation in fluctuating asymmetry than the mesiodistal diameter (Figs. 1 and 2). Directional asymmetry had no significant influence on these differences in fluctuating asymmetry (Table 3).

*TABLE 2. Summary statistics of rescaled asymmetry (d*) for MD and BL dimensions (categories pooled)*

| Tooth | Dimension | Male n | Male d* | Female n | Female d* | Difference *P* |
|---|---|---|---|---|---|---|
| I1 | MD | 202 | 0.006 | 219 | 0.005 | 0.467 |
| | BL | 202 | 0.009 | 221 | 0.008 | 0.675 |
| C | MD | 206 | 0.007 | 216 | 0.008 | 0.884 |
| | BL | 204 | 0.009 | 220 | 0.009 | 0.899 |
| M1 | MD | 206 | 0.004 | 210 | 0.005 | 0.436 |
| | BL | 202 | 0.007 | 214 | 0.006 | 0.606 |

d* = [R − L]/[R + L/2]. Other abbreviations as in Table 1.

An analysis of variance, testing for differences in gender, body mass, smoking status and interactions between the latter two (Table 4) confirmed that the combination of obesity and smoking status was a significant predictor of fluctuating asymmetry.

## DISCUSSION

Although there appears to be general agreement that obesity may be a risk factor to the mother during her pregnancy, the effects of maternal obesity on fetal development remain controversial. While some au-

*TABLE 3. Mean asymmetries for each of the four groups of children (genders pooled)*

| Tooth | Dimension | | Maternal smoking group: Obese smokers | Obese non-smokers | Non-obese smokers | Non-obese non-smokers[1] |
|---|---|---|---|---|---|---|
| I1 | MD | FA | 0.008** | 0.009** | 0.003 | 0.002 |
| | | DA | −0.001 | 0.000 | 0.000 | −0.001 |
| | BL | FA | 0.018** | 0.016** | 0.003 | 0.004 |
| | | DA | 0.001 | 0.000 | 0.001 | 0.000 |
| C | MD | FA | 0.007** | 0.006* | 0.002 | 0.004 |
| | | DA | 0.000 | −0.001 | 0.000 | 0.000 |
| | BL | FA | 0.010** | 0.010** | 0.005 | 0.006 |
| | | DA | 0.001 | 0.000 | 0.001 | 0.001 |
| M1 | MD | FA | 0.012** | 0.011* | 0.006 | 0.004 |
| | | DA | 0.001 | 0.000 | 0.001 | 0.000 |
| | BL | FA | 0.140** | 0.160** | 0.007 | 0.006 |
| | | DA | 0.000 | 0.000 | −0.001 | 0.000 |

FA = rescaled fluctuating asymmetry; DA = directional asymmetry; * $P < 0.005$; ** $P < 0.01$; significance of difference from status 4, (non-obese, non-smokers) as measured by Student's *t*-tests. Other abbreviations as in Table 1.
[1]Control group.

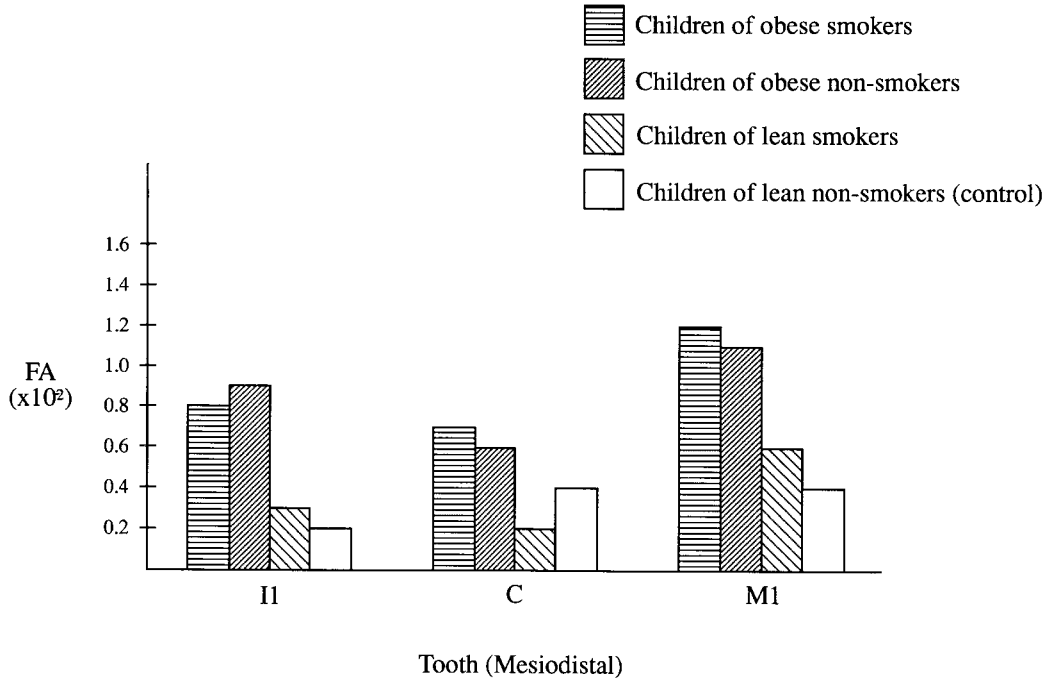


Fig. 1. Levels of fluctuating dental asymmetry (FA) in the children of lean and obese mothers—mesiodistal dimension. I1 = central maxillary incisor; C = maxillary canine; M1 = first maxillary molar.

thors have documented increased frequencies of miscarriage and perinatal mortality in obese mothers (Lucas et al., 1988; Naeye, 1990; Hamilton-Fairley et al., 1992), others have not (Gross et al., 1980; Gross, 1983; Harrison et al., 1980; Tilton et al., 1988).

One of the major findings of the present investigation is that children of obese mothers have higher levels of fluctuating asymmetry than those who mothers are lean during the pregnancy concerned. If one accepts the usefulness of fluctuating dental asymmetry as an indirect measure of developmental

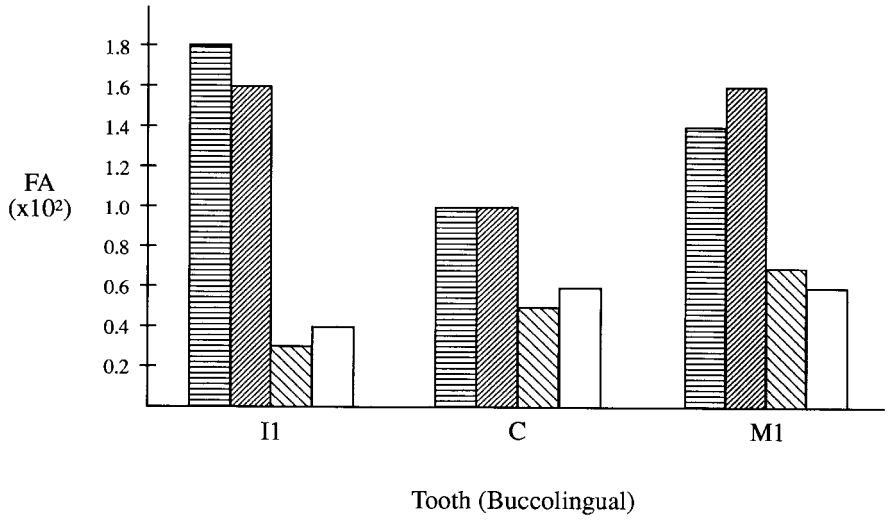


Fig. 2. Levels of fluctuating dental asymmetry (FA) in the children of lean and obese mothers—buccolingual dimension. For definition of columns, see Figure 1.

*TABLE 4. Results of an ANOVA testing for differences in fluctuating asymmetry due to gender, body mass, smoking and interactions between mass and smoking*

| Tooth | Source of variation | Mesiodistal | | | Buccolingual | | |
|---|---|---|---|---|---|---|---|
| | | DF | F | *P* | DF | F | *P* |
| I1 | Gender | 1 | 1.56 | 0.234 | 1 | 1.27 | 0.299 |
| | Mass | 1 | 0.41 | 0.568 | 1 | 0.68 | 0.763 |
| | Smoking status | 3 | 2.01 | 0.105 | 3 | 2.14 | 0.099 |
| | Mass × smoking | 3 | 4.38 | 0.010* | 3 | 4.73 | 0.014* |
| C | Gender | 1 | 1.94 | 0.137 | 1 | 2.19 | 0.257 |
| | Mass | 1 | 0.81 | 0.503 | 1 | 0.73 | 0.548 |
| | Smoking status | 3 | 1.37 | 0.493 | 3 | 0.99 | 0.073 |
| | Mass × smoking | 3 | 5.05 | 0.007** | 3 | 6.11 | 0.006** |
| M1 | Gender | 1 | 2.14 | 0.117 | 1 | 1.93 | 0.242 |
| | Mass | 1 | 0.47 | 0.731 | 1 | 0.52 | 0.646 |
| | Smoking status | 3 | 0.86 | 0.551 | 3 | 0.72 | 0.439 |
| | Mass × smoking | 3 | 5.67 | 0.005** | 3 | 6.64 | 0.004** |

*$P < 0.05$; ** $P < 0.01$; DF = degrees of freedom; I1 = central maxillary incisor; C = maxillary canine; M1 = first maxillary molar.

destabilization (Kieser, 1990; Livshits and Kobyliansky, 1991; Zakharov, 1992), then it follows that maternal obesity appears to upset the developmental homeostasis of the fetus.

That obese mothers may have an altered metabolic milieu which is somehow stressful to fetal development was suggested by Lucas et al. (1988). It appears that there are two independent maternal nutritional factors that bear heavily on fetal development: first, the weight of the mother before the pregnancy and second, maternal weight gain during pregnancy (Kliegman and Gross, 1985). While the first relates to the prior nutritional status of the mother, the second may be seen as a gross reflection of nutritional status during the pregnancy. Numerous studies have shown that the infants of overweight mothers are larger (macrosomic) than the infants of lean mothers (Harrison et al., 1980; Kalkhoff et al., 1988; Tilton et al., 1988). In contrast, it has been shown that between 10 and 40% of obese pregnant women actually lose weight during pregnancy (Kliegman and Gross, 1985). Diminished maternal weight gain during pregnancy, coupled to augmented fetal growth,

suggests an imbalance of the maternal-fetal nutritional flux towards the developing fetus. Whether maternal mobilization of stored fuel is sufficient to meet the metabolic needs of an obese mother and her macrosomic fetus remains unclear. What is clear, however, is that children of obese mothers have significantly elevated levels of fluctuating dental asymmetry, which in turn suggests subclinical destabilization of the fetus.

It is of interest that gestational diabetes appears to be related to maternal obesity (Tilton et al., 1988). In a study of fluctuating dental asymmetry in the fetuses of diabetic rhesus macaques, Kohn and Bennett (1986) found significantly elevated levels of fluctuating asymmetry when compared to normal fetuses. This points to another possible cause of the subclinical destabilization observed in our study.

The second major finding of this investigation is that maternal smoking is associated with significant elevation in fluctuating asymmetry in obese women (Table 3, Figs. 1 and 2). Moreover, an analysis of variance suggests that in all the teeth examined, there is a significant interaction between maternal mass and smoking status (Table 4). The effects of maternal smoking on the developing fetus were controversial and were recently reviewed by Kieser and Groeneveld (1994). In contrast to that study, which reported no elevation of dental asymmetry in the children of smokers, we found that children of obese women who smoked during the pregnancy concerned had raised levels of fluctuating asymmetry. Whether this observation is due to the effect of tobacco smoke on the child or a combination of smoking and nutritionally related destabilization is unclear and deserves further investigation.

Finally, while some authors have reported no differences in the magnitudes of fluctuating asymmetry for MD and BL dimensions (e.g. Harris and Nweeia, 1980; Hershkovitz et al., 1993), others have found that the MD dimension showed significantly more asymmetry than the BL dimension (e.g. Townsend and Brown, 1980). Whether the finding here that MD asymmetry values were lower than those of BL dimensions suggests independence of these two dimensions is a question left unanswered.

## ACKNOWLEDGMENT

We thank the late Elli Lasersohn who collected the casts used in this study and to whose memory this paper is dedicated.

## LITERATURE CITED

Butler PM (1983) Evolution and mammalian dental morphology. J. Biol. Buccale *11:*285–302.

DiBennardo R, and Bailit HL (1978) Stress and dental asymmetry in a population of Japanese children. Am. J. Phys. Anthropol. *48:*89–94.

Douglas GW, and Scadron EN (1951) The influence of obesity in pregnancy. Med. Clin. North Am *35:*733–736.

Fraser FC (1994) Developmental instability and fluctuating asymmetry in man. In TA Markow (ed.): Developmental Instability: Its Origins and Evolutionary Implications. Amsterdam: Kluwer, pp 319–354.

Gest TR, Siegel MI, and Anistranski J (1982) Increased fluctuating asymmetry in the long bones of rats stressed by cold, heat and noise. Am. J. Phys. Anthropol. *62:*196–199.

Gross TL (1983) Operative considerations in the obese pregnant patient. Clin. Perinatol. *10:*411–421.

Gross TL, Sokol RJ, and King KC (1980) Obesity in pregnancy: Risks and outcomes. Obstet. Gynecol. *56:*446–450.

Hamilton-Fairley D, Kiddy D, Watson H, Paterson C, and Franks S (1992) Association of moderate obesity with a poor pregnancy outcome in women with polycystic ovary syndrome treated with low dose gonadotrophins. Br. J. Obstet. Gynaecol. *99:*128–131.

Harris EF, and Nweeia M (1980) Dental asymmetry as a measure of stress in the Ticuna Indians of Colombia. Am. J. Phys. Anthropol. *53:*133–142.

Harrison GG, Udall JN, and Morrow PG (1980) Maternal obesity, weight gain in pregnancy and infant birth weight. Am. J. Obstet. Gynecol. *136:*410–411.

Hershkovitz I, Livshits G, Moskona D, Arensburg B, and Kobyliansky E (1993) Variables affecting dental fluctuating asymmetry in human isolates. Am. J. Phys. Anthropol. *91:*349–365.

Kalkof RK, Kandaraki PG, Morrow PG, Mitchell TH, Kelber S, and Borkowf HI (1988) Relationship between neonatal birth weight and maternal plasma amino acid profiles in lean and obese nondiabetic women. Metabolism *37:*234–239.

Kieser JA (1990) Human Adult Odontometrics. Cambridge: Cambridge University Press.

Kieser JA, and Groeneveld HT (1988) Fluctuating odontometric asymmetry in an urban South African black populations. J. Dent. Res. *69:*1200–1205.

Kieser JA, and Groeneveld HT (1994) Effects of prenatal exposure to tobacco smoke on developmental stability in children. J. Craniofac. Genet. Dev. Biol. *14:*43–47.

Kieser JA, Groeneveld HT, and Preston CB (1986) Fluctuating odontometric asymmetry in the Lengua Indians of Paraguay. Ann. Hum. Biol. *13:*489–498.

Kieser JA, Groeneveld HT, McKee J, and Cameron N (1990) Measurement error in human dental mensuration. Ann. Hum. Biol. *17:*523–528.

Kliegman RM, and Gross T (1985) Perinatal problems of the obese mother and her infant. Obstet. Gynecol. *66:*299–305.

Kohn LA, and Bennett KA (1986) Fluctuating asymmetry in fetuses of diabetic rhesus macaques. Am. J. Phys. Anthropol. *71:*477–483.

Livshits G, and Kobyliansky E (1989) Decreased developmental stability as assessed by fluctuating asymmetry of morphometric traits in preterm infants. Am. J. Med. Genet. *19:*793–805.

Livshits G, and Kobyliansky E (1991) Fluctuating asymmetry as a possible measure of developmental homeostasis in humans: A review. Hum. Biol. *63:*441–466.

Longcope C, Baker R, and Johnston CC (1986) Androgen and estrogen metabolism: Relationship to obesity. Metabolism *35:*235–237.

Lucas A, Morley R, Cole TJ, Bamford MF, Boon A, Crowle P, Dossetor JFB, and Pearse R (1988) Maternal fatness and viability of preterm infants. Br. Med. J. *296:*1495–1497.

Mathews HB, and Der Funcke MG (1938) Normal expectancy in the extremely obese pregnant woman. J. Am. Med. Assoc. *11:*554–555.

Mooney MP, Siegel MI, and Gest TR (1985) Prenatal stress and increased fluctuating asymmetry in the parietal bones of neonatal rats. Am. J. Phys. Anthropol. *68:*131–134.

Naeye RL (1990) Maternal body weight and pregnancy outcome. Am. J. Clin. Nutr. *52:*273–279.

Parsons PA (1990) Fluctuating asymmetry: An epigenetic measure of stress. Biol. Rev. *65:*131–145.

Palmer AR, and Strobeck C (1986) Fluctuating asymmetry: Measurement, analysis, patterns. Ann. Rev. Ecol. Syst. *17:*391–421.

Petry JA (1956) Obesity with pregnancy. Obstet. Gynecol. *7:*299–302.

Siegel MI, and Doyle WJ (1975) The effects of cold stress on fluctuating asymmetry in the dentition of the mouse. J. Exp. Zool. *93:*385–389.

Sofaer JA (1979) Human tooth-size asymmetry in cleft lip with or without cleft palate. Arch. Oral Biol. *24:*141–146.

Suarez BT (1974) Neanderthal dental asymmetry and the probable mutation effect. Am. J. Phys. Anthropol. *41:*411–416.

Tilton Z, Hodgson MI, Donoso E, Areaga A, and Rosso P (1988) Complications and outcome of pregnancy in obese women. Nutrition *5:*95–99.

Townsend GC (1981) Fluctuating dental asymmetry in the deciduous dentition of Australian Aborigines. J. Dent. Res. *60:*1849–1857.

Townsend GC (1983) Fluctuating dental asymmetry in Down syndrome. Aust. Dent. J. *28:*39–44.

Townsend GC, and Brown T (1980) Dental asymmetry in Australian Aboriginals. Hum. Biol. *52:*661–673.

Townsend GC, Alvesalo L, Jensen B, and Kari M (1986) Patterns of tooth size in human chromosomal aneuploidies. Mem Mus Nat. Hist. Paris *53:*25–45.

Van Valen L (1962) A study of fluctuating asymmetry. Evolution *16:*125–142.

Witten SB (1958) Labor in the obese patient. Obstet. Gynecol. *12:*99–104.

Zakharov VM (1992) Population phenogenetics: Analysis of developmental stability in natural populations. Acta Zool. Fenn. *191:*7–30.